Armed Forces College of Medicine

AFCM

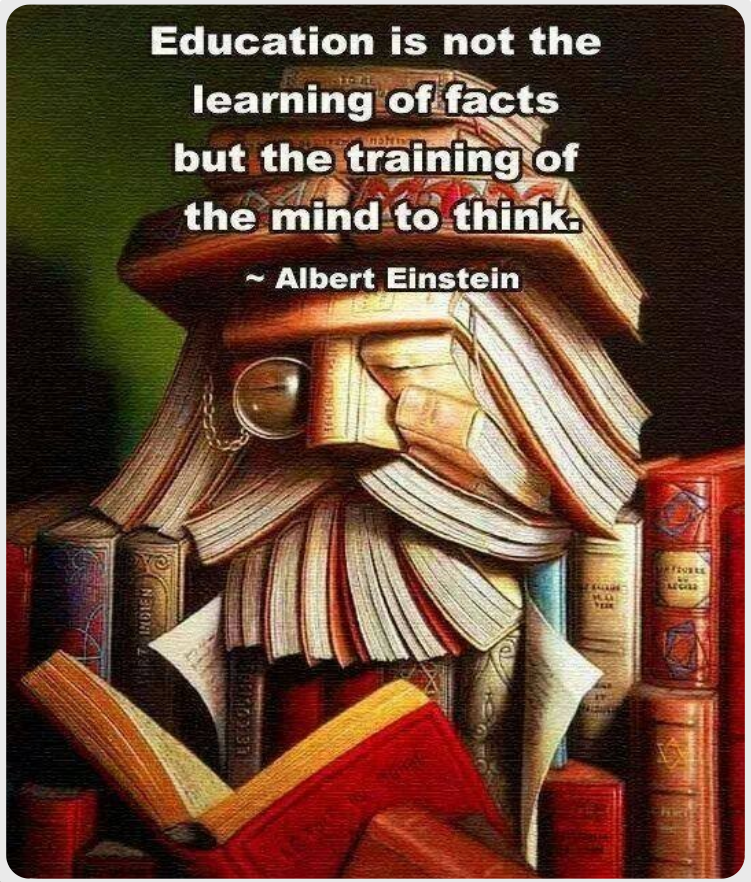
Cardiopulmonary module

Clinical Integrated Cases

Session 4

Basic Science Principles for Clinical Reasoning

1- Critical thinking
2- Introduction to common forms of disease
3- Apply basic knowledge in a realistic clinical scenario.
4- Interpersonal skills-(share and apply your knowledge)… communication…patient safety
5- Life long self-directed learning



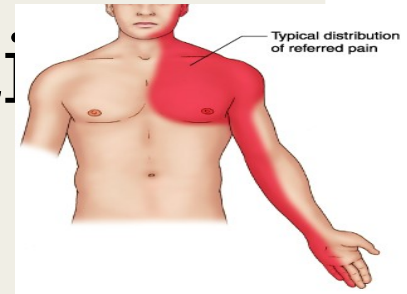
Case co-ordinators

Prof. Drs. Manal Hassan, Tamer Wasify, lamiaa, Abeer

Case: I have a chest pain

I have a chest pain

Adel is a **59** years old **obese** man. He ***smokes*** 30 cigarettes/day.
He has been admitted to the emergency room with 90-minute history of severe ***crushing chest pain, chest tightness*** that was **retrosternal** radiati
the left shoulder
upper arm.



I have a chest pain

On his way to the hospital, Adel took **sublingual nitroglycerine** with ***no*** prominent relief.

The physician asked Adel about past history of attacks of ***chest pain***. Adel said that he had chest pain **on exertion** that was **relieved by rest and sublingual nitroglycerine.**

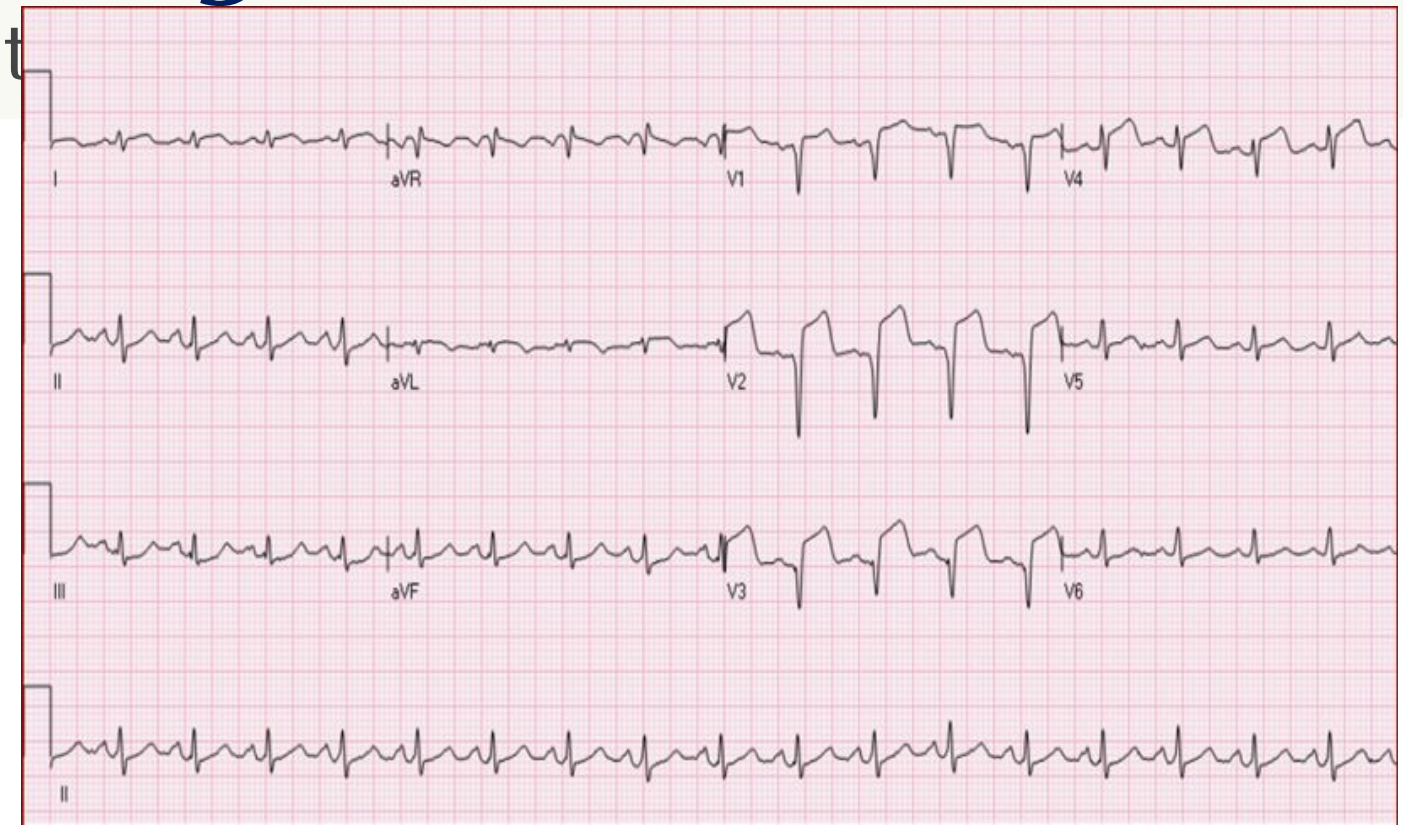
I have a chest pain

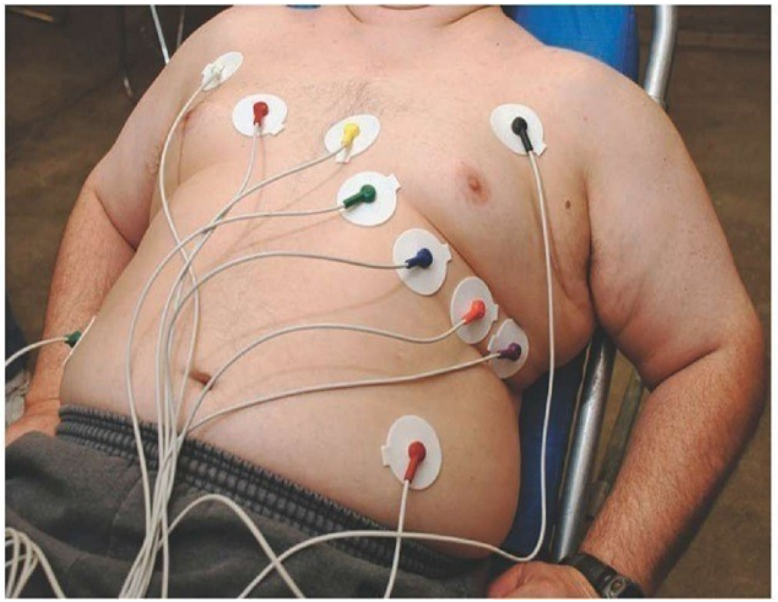
His blood pressure was **150/90** mm Hg

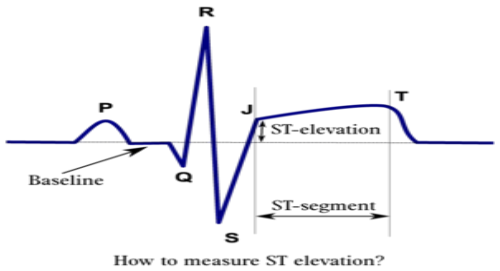
An emergency **ECG** was done.

I have a chest pain

His ***ECG (done within 10 minutes of arrival)*** showed 3mm ***ST segment elevation*** in leads V_1 t







How to measure ST elevation?

Obese, *complain of crushing chest pain*.

1- What is the differential diagnosis of acute chest pain in Adel?

Justify your answer

DIFFERENTIAL DIAGNOSIS OF CHEST PAIN IN Adel:

1- Acute coronary syndrome due to the typical chest pain and the presence of many risk factors for atherosclerosis including: male sex, age , obesity, smoking, dyslipidemia, hypertension and the history of typical anginal pain on exertion.

2- Dissecting aortic aneurysm

3-pulmonary embolism,

4- Pericarditis.

2- Mention the risk factors in this case

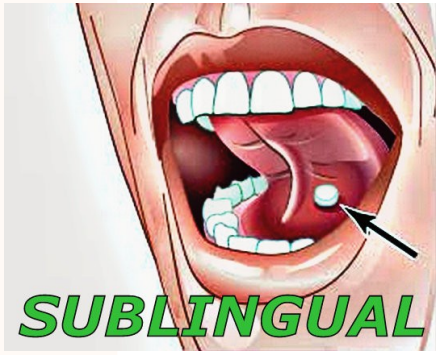
- Male gender
- Age (men >45, women >55 years)
- Obesity
- Smoking
- Hypertension??
- Dyslipidemia??

The physician asked about his past history of attacks of chest pain. Adel said that he had ***chest pain* on exertion** that was **relieved by rest and sublingual nitroglycerine**.

Analyze the symptom of chest pain on exertion in Adel

Adel took **sublingual nitroglycerine** with ***no*** prominent relief

3- a-Why did Adel take sublingual nitroglycerine?



b- What is the effect of nitroglycerine?

Why did Adel take sublingual nitroglycerine?

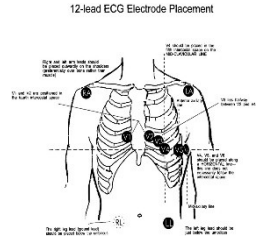
For immediate relieve of ischemic chest pain

Effect of nitroglycerine

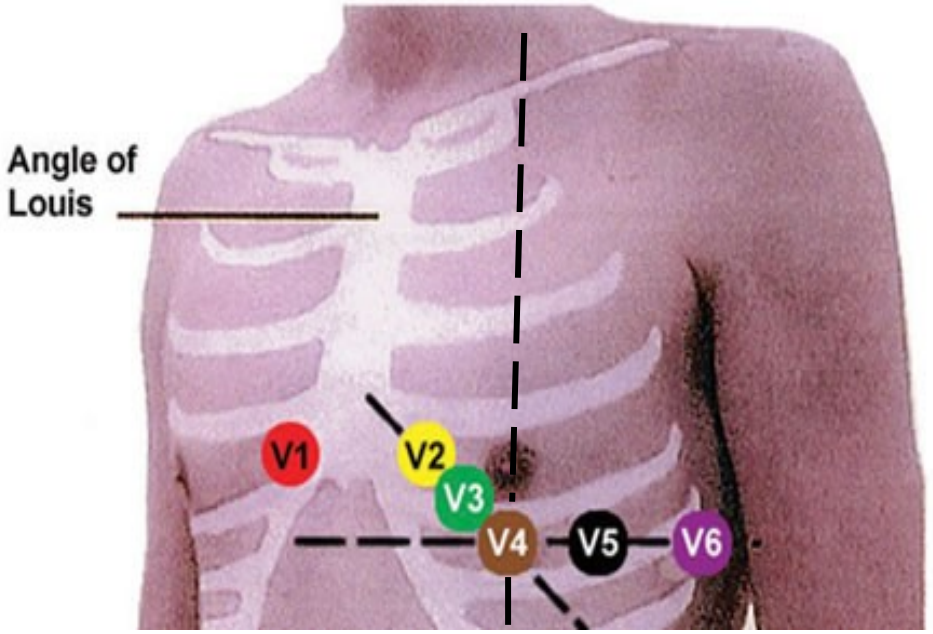
1. It causes dilation of the large veins, resulting in pooling of blood in the veins. This diminishes preload (venous return to the heart) reduces the work of the heart.
2. *Nitroglycerin* dilates the coronary vasculature, providing an increased blood supply to the heart muscle.
3. *Nitroglycerin decreases myocardial oxygen* consumption because of decreased cardiac

An emergency ECG was done.

4- How to place the leads of ECG to Adel?



Chest leads



V1: 4th right intercostal space parasternal

V2: 4th left intercostal space parasternal

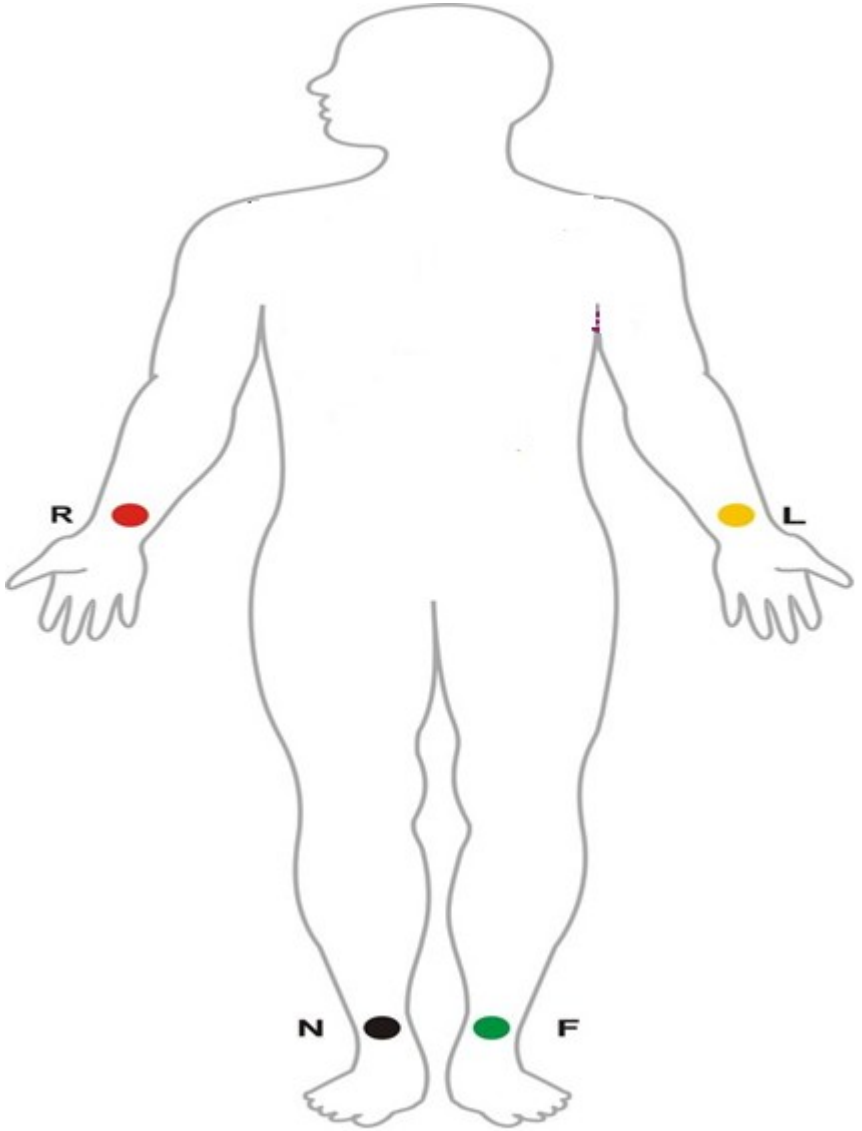
V3: midway between V2 & V4

V4: 5th left intercostal space mid-clavicular line

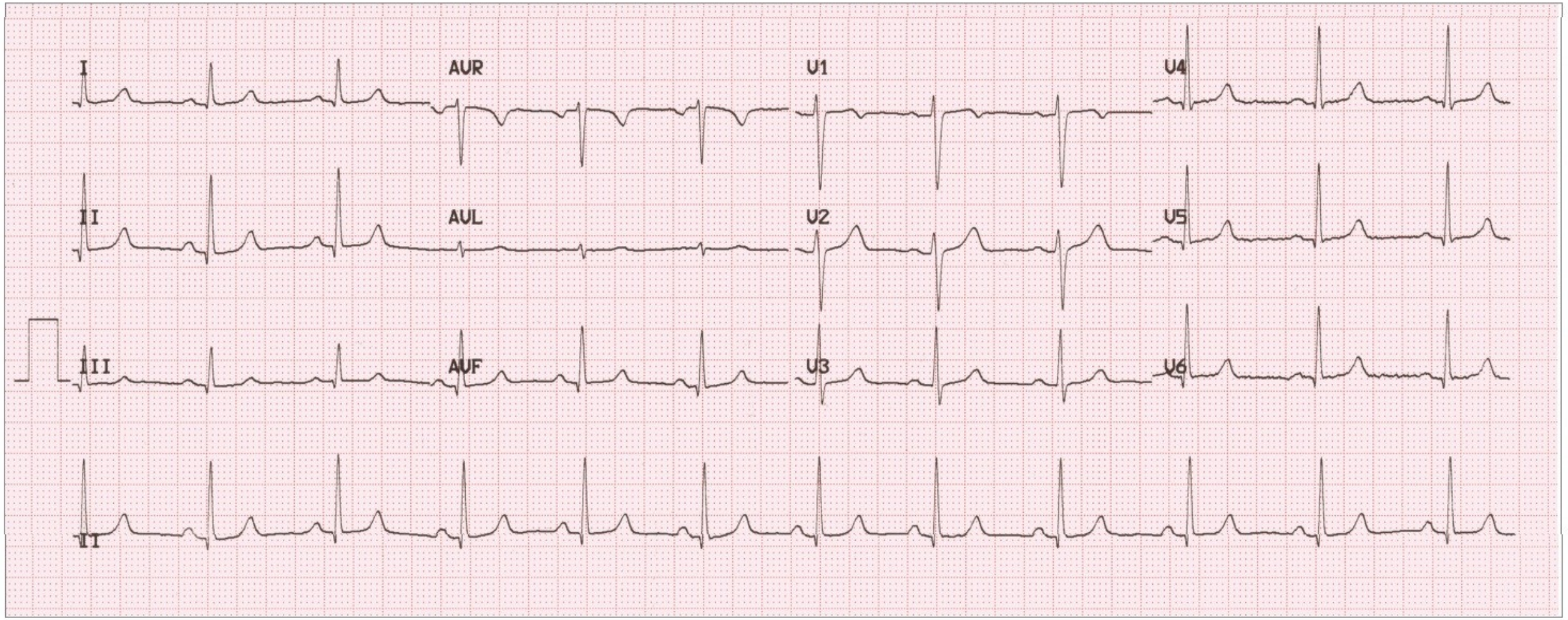
V5: At the Same level as V4 left anterior axillary line

V6: At the Same level as V4 left mid-axillary line

Limb leads

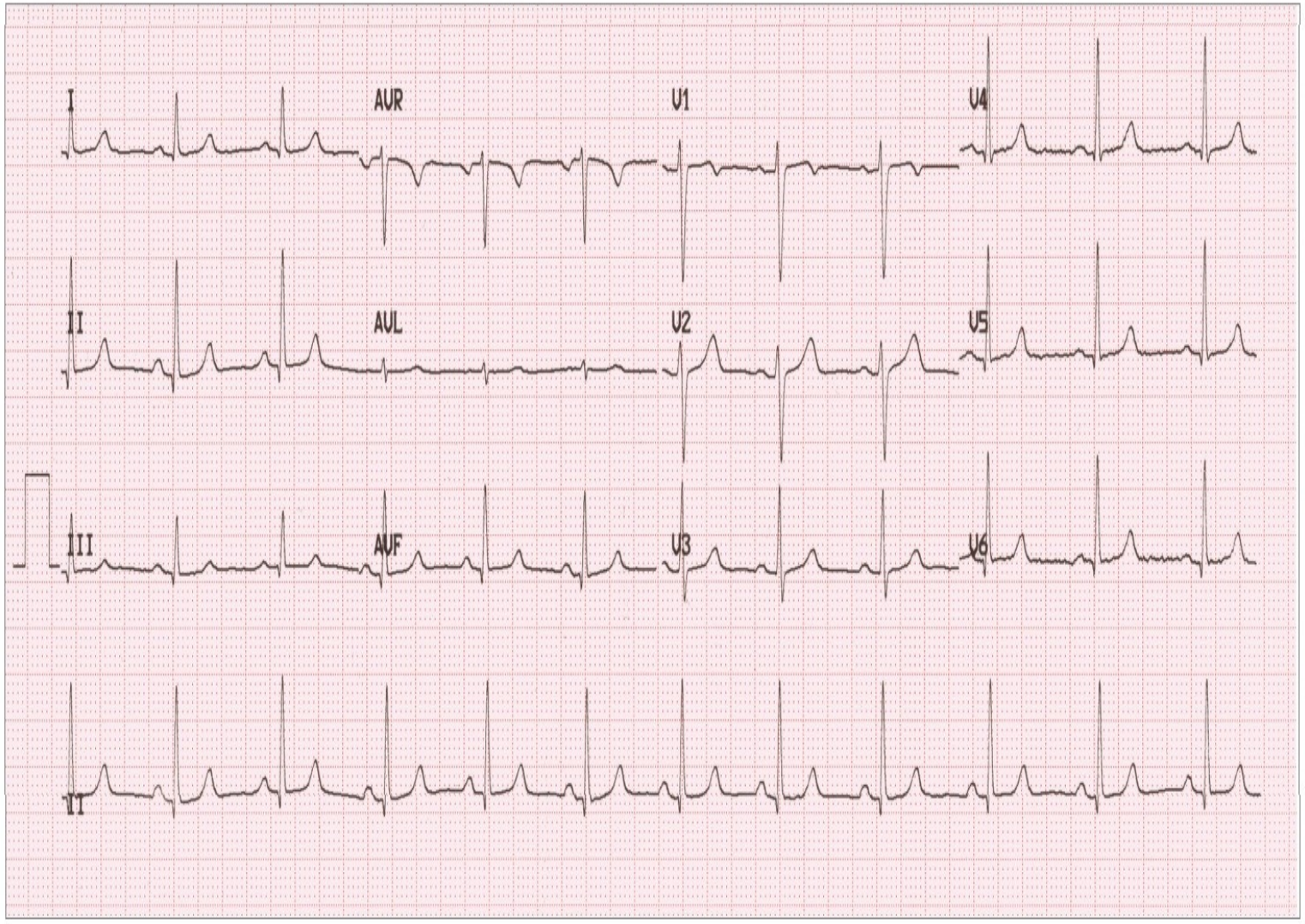


5- What are the normal ECG patterns?

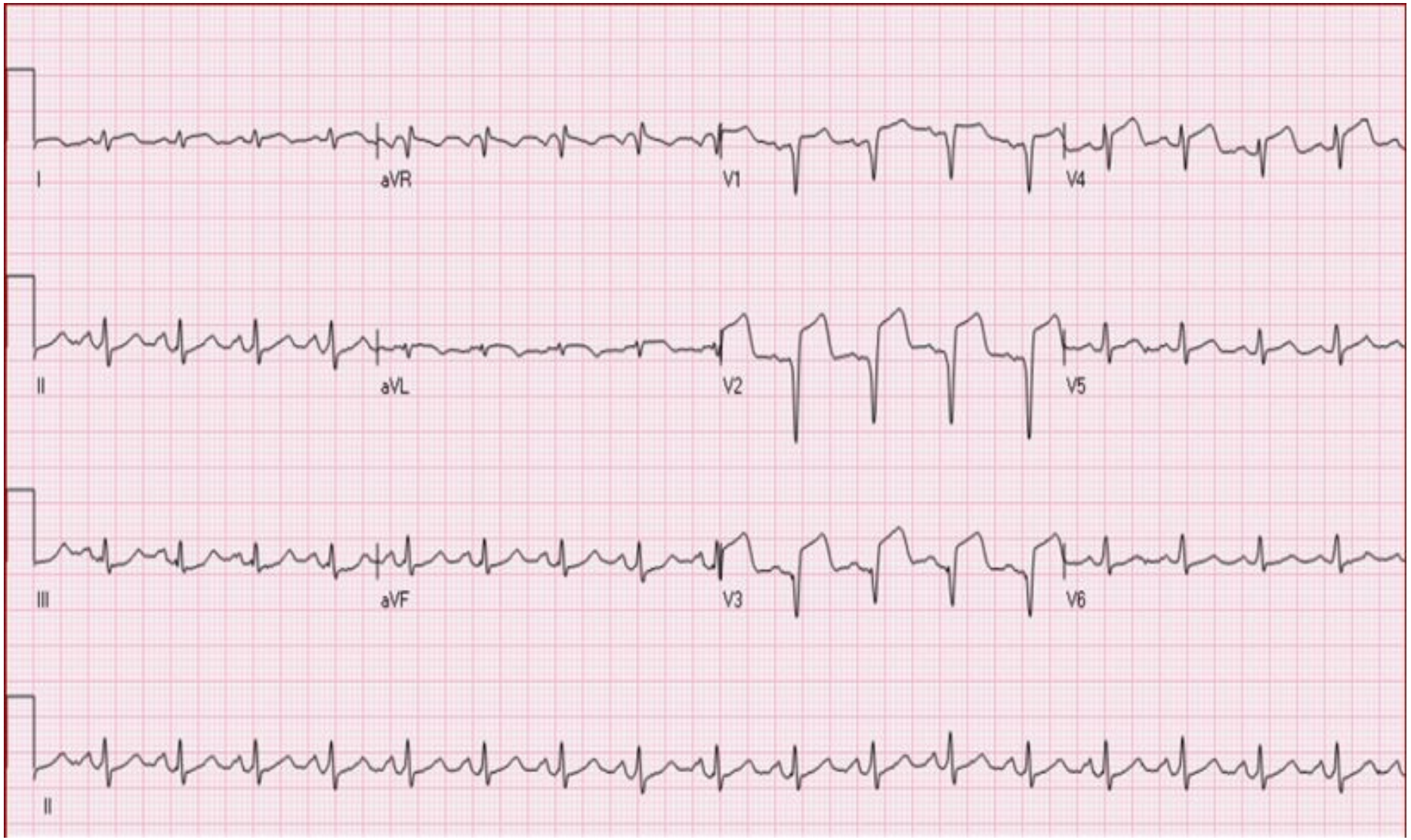


Normal sinus rhythm

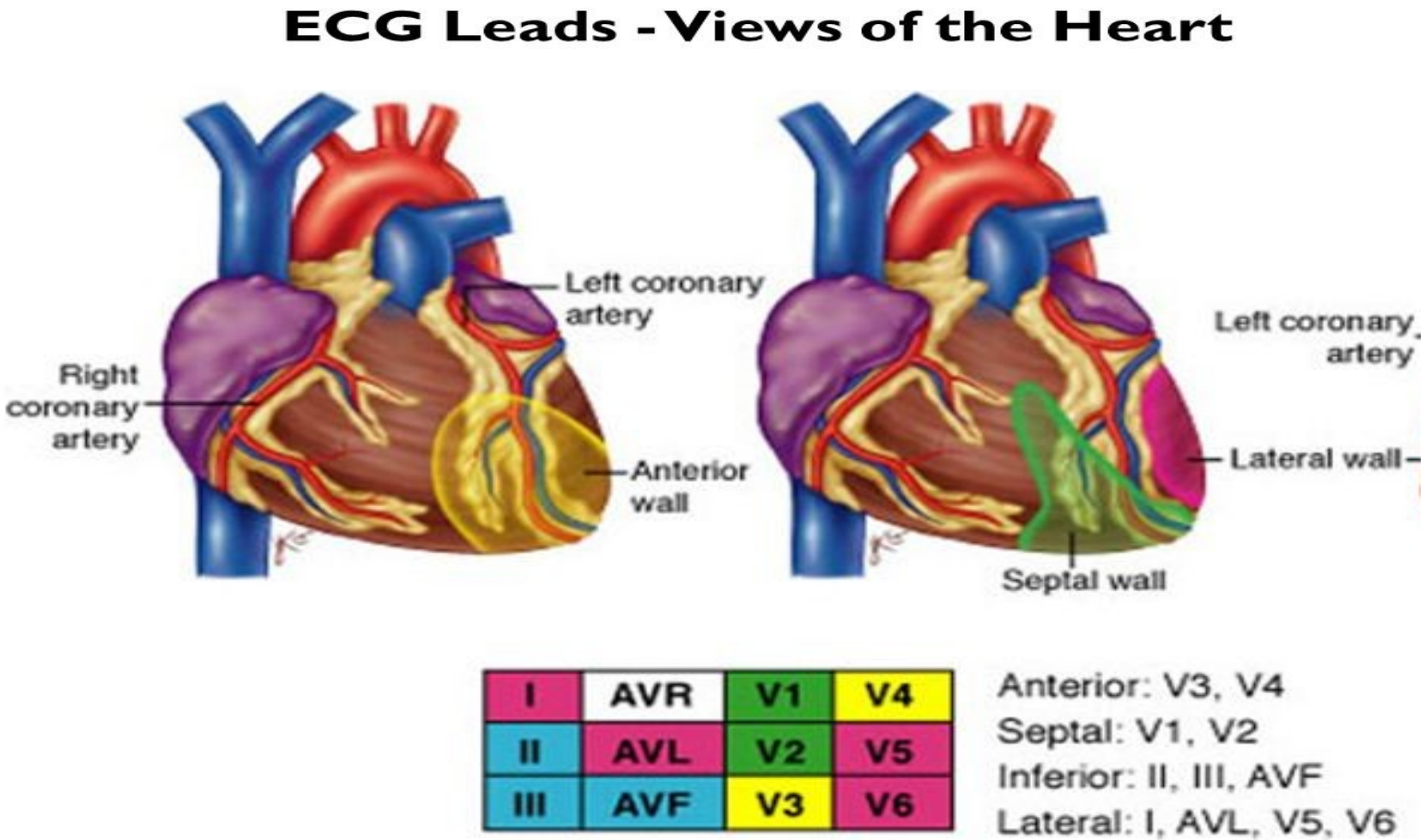
- **Rhythm: Regular**
- **Heart rate: 60-100 beat/min**
- **Waves:**
  - **Every P wave followed by QRS complex**
  - **P wave, QRS complex and T wave of normal duration, amplitude and polarity *(waves in aVR in opposite direction to LII e.g. P wave positive in lead II and negative in aVR)***
  - **T wave in same direction as QRS complex**
- **Intervals:**
  - **PR interval = 0.12-0.21 sec**
  - **QT interval = 0.36-0.4**



This was Adel’s ECG. What is the abnormality detected?



His ***ECG*** showed 3mm **ST segment elevation** in leads V_1 to V_4 consistent with an **acute anterior myocardial infarction.**



Describe the blood supply of the anterior wall of heart and interventricular septum?

Right Coronary artery:

Arises from anterior aortic sinus of ascending aorta.

It supplies the anterior wall of heart through the following branches:

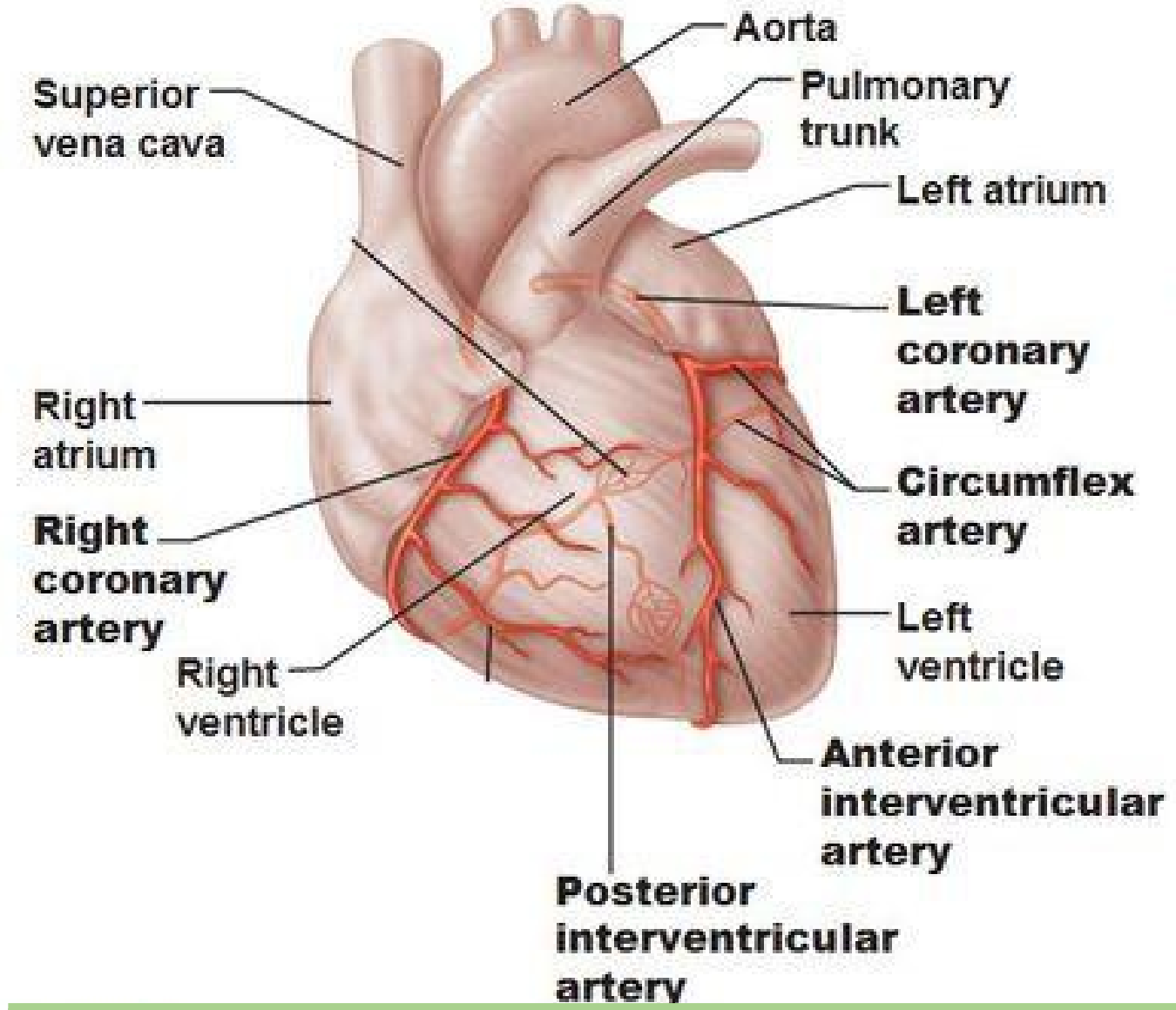
1) Right conus artery infundibulum of right ventricle
2) Right marginal artery right ventricle
3) Atrial & ventricular branches both atria & right ventricle

Left Coronary artery:

Arises from left posterior aortic sinus of ascending aorta.

It supplies the anterior wall of the heart through the following branches:

1) Anterior interventricular (LAD) Adjacent parts of both ventricles
2) Circumflex artery Atrial & ventricular brs to left atrium & left ventricle



Interventricular septum is supplied by

Anterior ⅔ LAD (anterior interventricular artery)

Posterior ⅓ RPD (Posterior interventricular artery)

6- Deduce which artery is affected in Adel?

Justify your answer

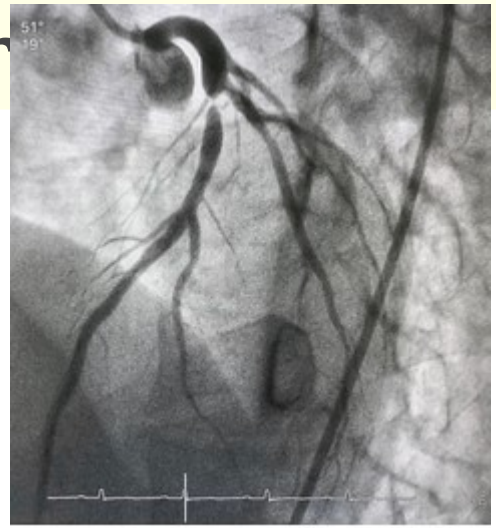
Left anterior descending branch of the left coronary artery because the patient has **acute anterior myocardial infarction**

Welcome back

I have a chest pain

Adel was given loading doses of ***aspirin*** and ***clopidogrel***.

Forty
he underwent successful primary **percutaneous coronary intervention** ***(PCI)*** with the insertion of a drug eluting stent into his critically narrowed **coronary arter**



Chest Pain

Adel was returned to the coronary care unit after PCI, he was pain free and there was **partial resolution of his ECG changes.**

The doctor took a detailed history, Adel has been **hypertensive for 20 years** but did **not** take regular therapy.

The doctor asked for **several investigations:**

1- Cardiac Markers

Cardiac ***troponins*** and **CK-MB** were elevated

Chest Pain

2- Fasting lipid profile:

Cholesterol = **320**mg/dl;

HDL cholesterol = **20**mg/dl;

LDL cholesterol = **190**mg/dl;

Triglycerides = **240**mg/dl

3- CBC : normal

4- Liver Enzymes and kidney function: normal

5- HBA1c: normal

6- Echocardiography -----

to assess cardiac function

He was given loading doses of ***aspirin*** and ***clopidogrel***.

7- Why was Adel given aspirin and clopidogrel before doing PCI?

What is the physiological effect of aspirin and clopidogrel?

They both have antiplatelet effect

For aspirin it acts through Irreversible inhibition of Platelet Thromboxane Synthetase enzyme TXA2 (COX enzyme)

For clopidogrel it blocks the ADP receptors on the platelets thus inhibiting platelet aggregation.

After admission, he underwent ***percutaneous coronary intervention (PCI)*** with insertion of a drug eluting stent into his narrowed left anterior descending coronary artery.

8- What is PCI?

What is the role of PCI in myocardial infarction?

Percutaneous Coronary Intervention

Primary PCI with opening of the artery in catheter lab and putting stent is preferred to thrombolytic therapy
If primary PCI is not available within less than 120 minutes from start of chest pain, proceed to thrombolytic therapy.

Why rapid PCI in treating myocardial infarction?

“Time is muscle”

By the time he was returned to the coronary care unit 30 minutes after the procedure, he was pain free and there was **partial resolution of his ECG changes**.

What is the normal histology of the cardiac muscle?

LM of Cardiac Muscle

1. Cylindrical in shape.
2. They are branching & anastomosing fibers.
3. They are smaller in diameter than skeletal ms.
4. Nucleus: single, oval & central.
5. The cytoplasm:-

- Moderately acidophilic.
- It shows less distinct striation in comparison to skeletal ms.??????

6. The cardiac m. cells are attached to each other by **Intercalated discs**→ results in **fibers** of variable length.

- **No satellite cells**

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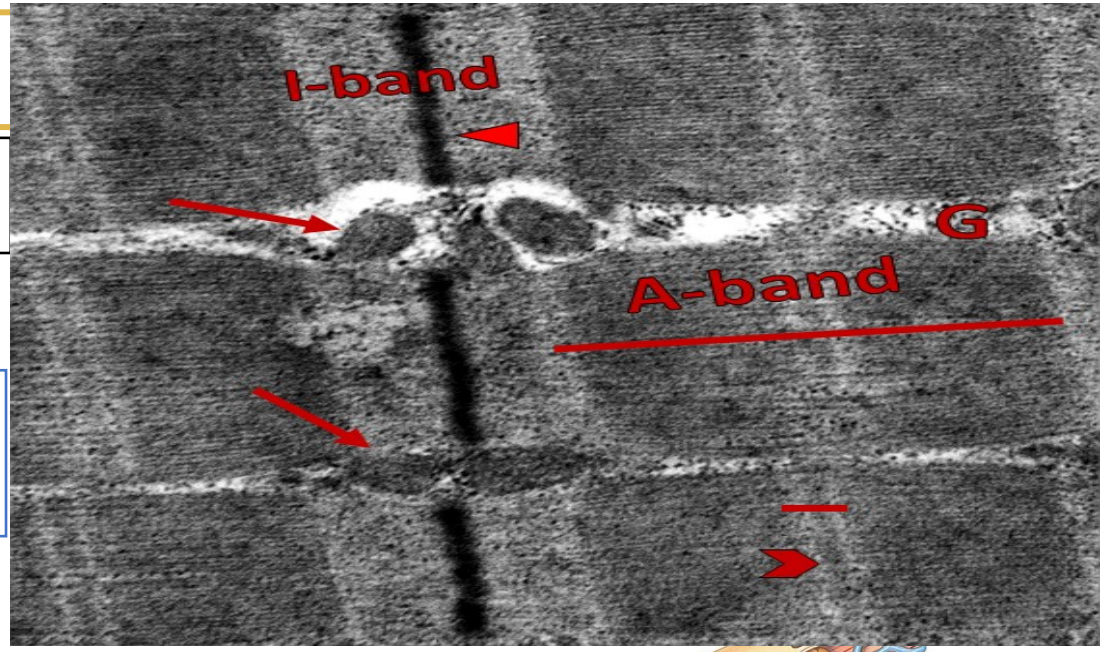
EM of Cardiac Muscle

1. Myofibrils & Myofilaments: sarcomere

2. Cardiac T –tubules: Wide, at Z

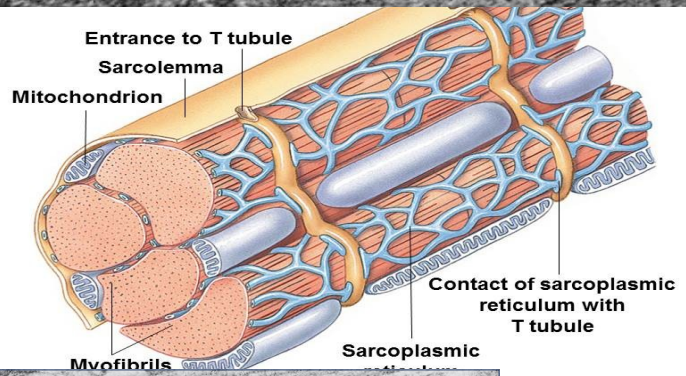
3. Sarcoplasmic Reticulum: forms single cisternae on one side of T-tubules→ diad.



4. Mitochondria: abundant (40%)

5. Lipid droplets & glycogen granules …..source of energy.

6. Myoglobin &: Lipofuscin pigment

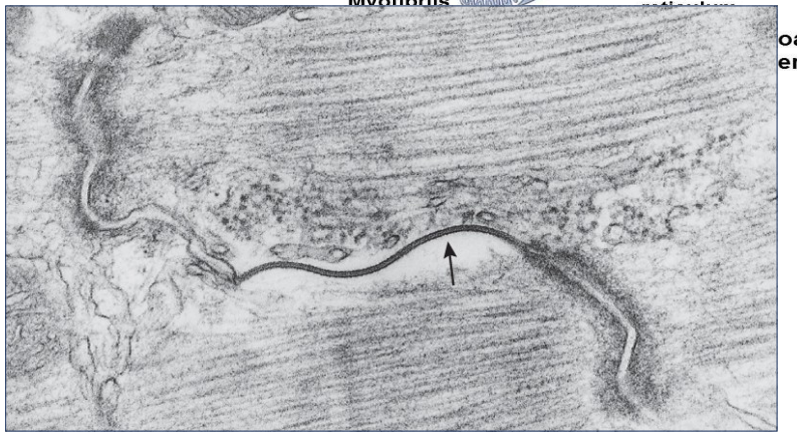


7. **Intercalated discs**: a step-like junction formed of:

a. Transverse portion, formed mainly of fascia adherens…..*mechanical syncytium.*

b. Lateral portions, formed mainly of gap junction……*electrical syncytium.*

- *Both of them are reinforced by desmosomes.*



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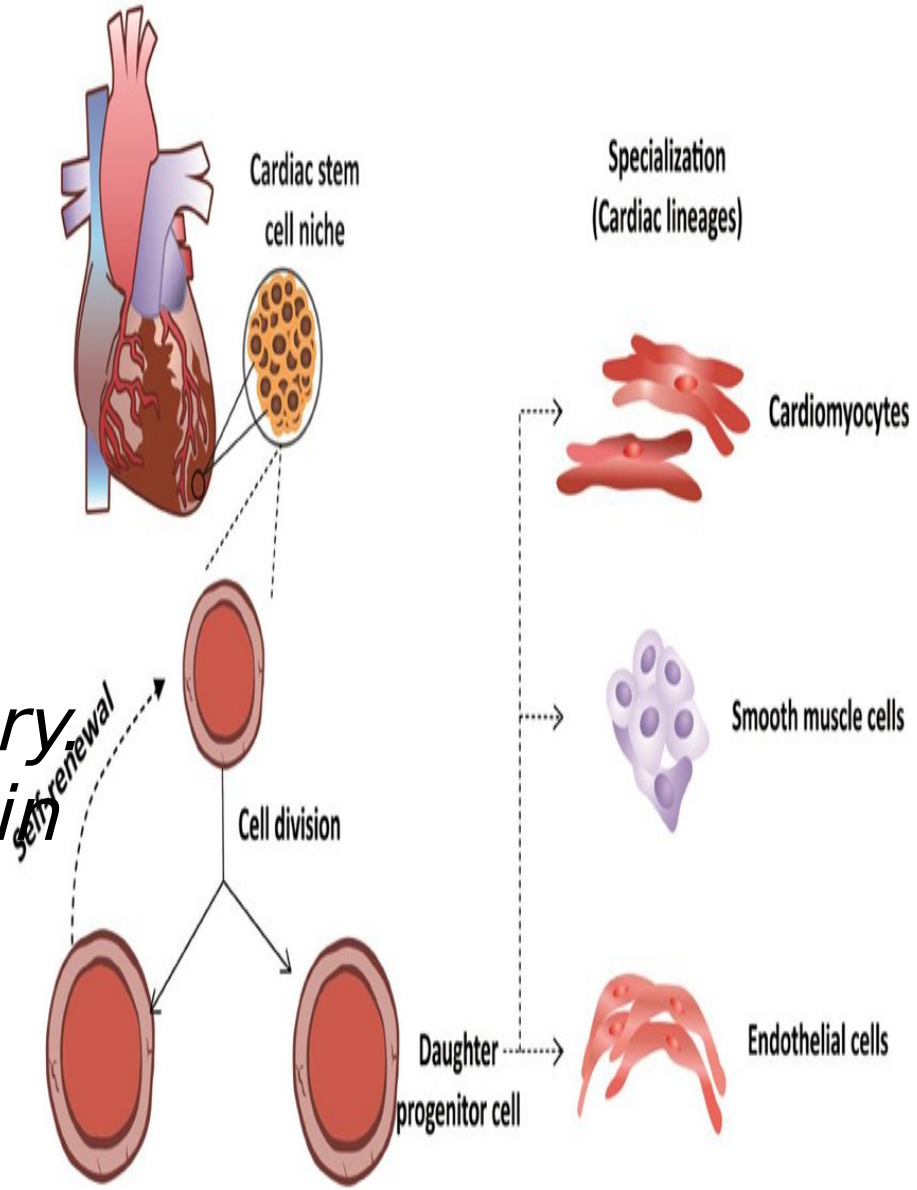
ernae.

Growth & Regeneration of Cardiac Muscle

- Cardiac muscle can`t divide and devoid of satellite cells…… **No regeneration.**

- **Healing of injured myocardium occurs by fibrosis.** *Consequently, cardiac function is lost at the site of injury. This pattern of injury and repair is seen in* ***myocardial infarction (M I).***



Cardiac troponins and CPK were mildly elevated

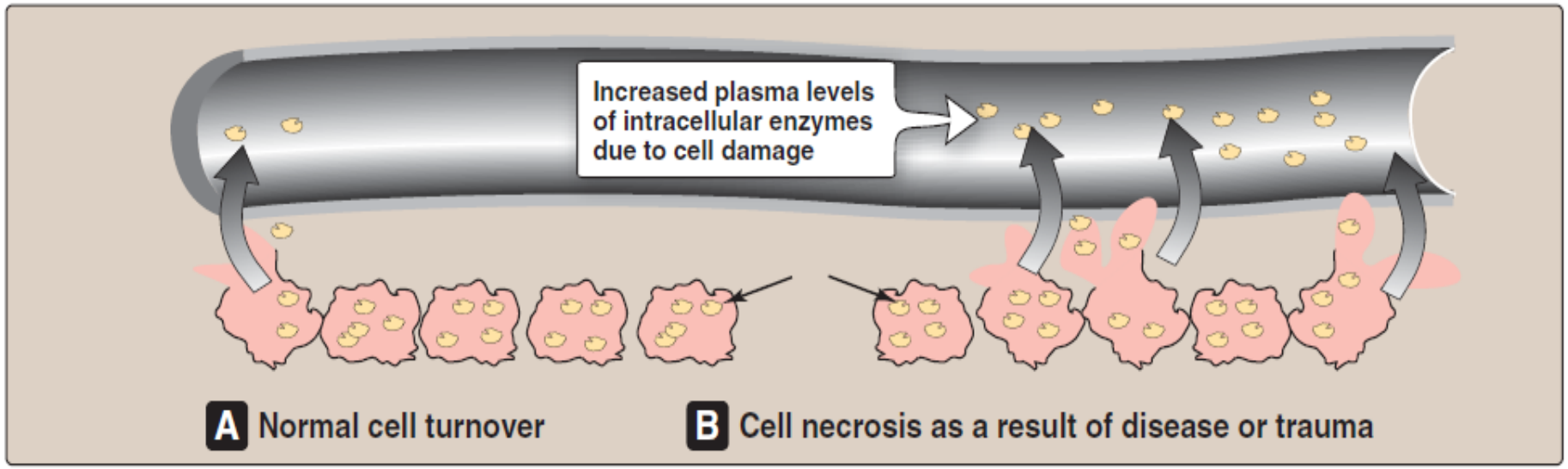
9- Interpret the increase in cardiac troponins and CK-MB?

Biochemical changes in MI

Death of cardiomyocytes ⟶

abnormal release of the cardiac markers (proteins "mostly enzymes" which are normally present inside the cardiomyocytes) into circulation ⟶

abnormal increased levels in blood


Increased plasma levels of intracellular enzymes due to cell damage
A Normal cell turnover
B Cell necrosis as a result of disease or trauma

Time sequence of cardiac biomarkers changes with MI

Cardiac marker	Rises	Peaks	Returns to normal
Myoglobin	**1-2 hours**	**6-8 hours**	**1 day**
Troponin	**1-2 hours**	**1-2 days**	**3-5 days (TnI)** **5-14 days (TnT)**
CK2	**4-8 hours**	**24 hours**	**2-3 days**
LDH1	**8-12 hours**	**3-6 days**	**8-14 days**

Cardiac biomarkers changes with MI

- TnI & CK2 are used for early diagnosis of MI.
- Myoglobin is used for as a negative predictor of AMI.
- TnT & LDH1 are used for follow up of MI

Fasting lipid profile:
Cholesterol = 320mg/dl;
HDL cholesterol = 20mg/dl;
LDL cholesterol = 190mg/dl;
Triglycerides = 240mg/dl

Explain the importance of investigating the levels of cholesterol, LDL and HDL in Adel

Disorders of plasma lipoproteins

Hyperlipoproteinemia and Hypolipoproteinemia

Hyperlipidemia, hyperlipoproteinemia or dyslipidemia:

- is the presence of raised or abnormal levels of lipids and/or lipoproteins in the blood. The type of lipoprotein determines the fate of the particle and its influence on metabolism.
- Lipid and lipoprotein abnormalities are extremely common in the general population and are regarded as a highly risk factor for cardiovascular disease due to the influence of cholesterol, one of the most clinically relevant lipid substances, on atherosclerosis.

Lipid profile ..... why?

Because Adel has MI with risk factors of atherosclerosis so we chick hypercholestermia & dyslipidemia

Lipid profile	Normal	Adel
Total Cholesterol	< 200 mg/dl	**320mg/dl**
LDL-Cholesterol	< 100 mg/dl	**190mg/dL**
HDL-Cholesterol	≥ 60 mg/dl	**20mg/dl**
Triglycerides	< 150 mg/dl	**240mg/dl**

Biochemical basis of development of atherosclerosis

1- Modified LDL (oxLDL: oxidized lipids and apolipoprotein) due to oxidative stress

2- Uptake of oxLDL by macrophage scavenger receptor class A (SR-A) Foam cell transformation and atherosclerotic plaque formation

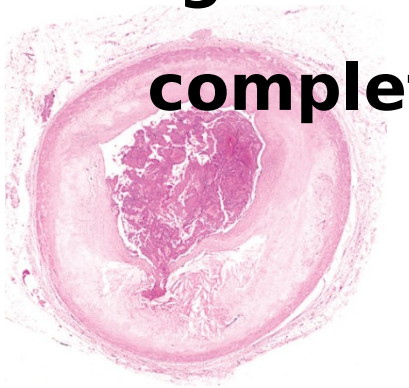
How could atherosclerosis lead to myocardial infarction?

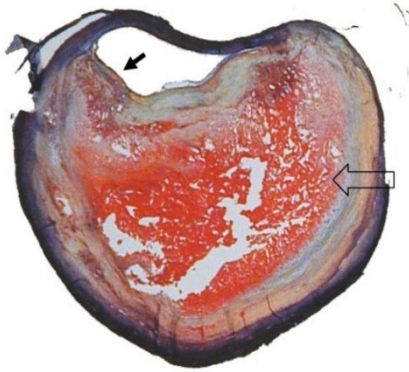
Causes:

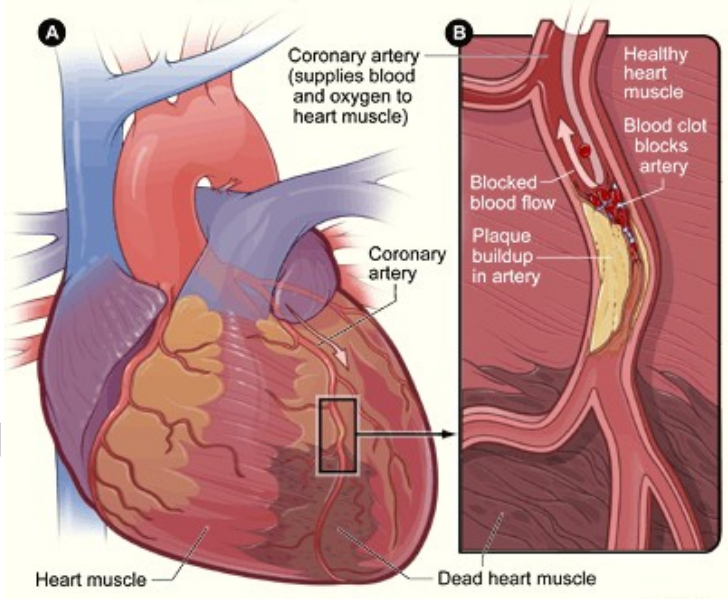
Coronary atherosclerosis + complete occlusion (90% of cases)

due to:

- **Advanced atherosclerosis**
- **Thrombosis over an atheroma →**
- **Intimal hemorrhage in an atheroma → lifting the atheroma → complete occ**







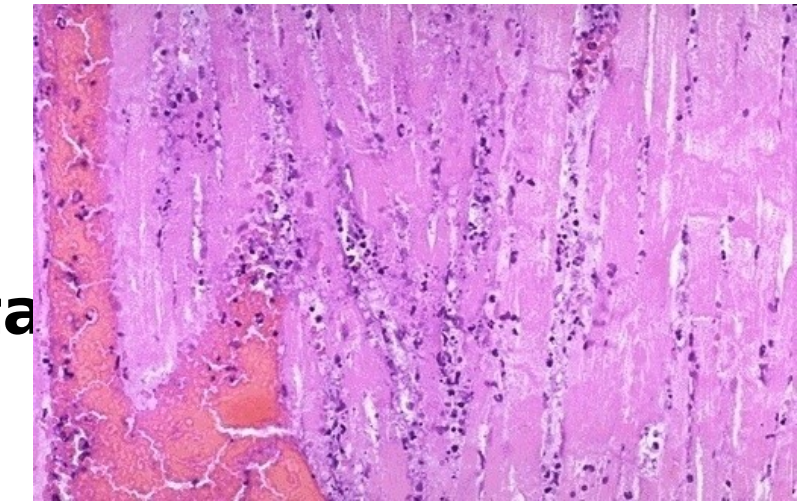
10- What is the pathological features and complications of acute myocardial infarction if not treated early?

icture:

ges appear after 6-8 h from onset of infarction
t: swollen, pale, friable, may rupture, hyperemic m
thrombus may occur
brinous pericarditis

opic picture:

ges appear earlier than gross changes
***ulative necrosis* Infiltrated by neutrophils followed by macrophages**



cations:

heart failure.
ure of infarct → hemopericardium & death
thmias: e.g. ventricular fibrillation., may be fata
due to cardiogenic shock.

Welcome back

Chest Pain

The doctor told Adel that he was lucky to receive early intervention as the complications would have been very seri

The doctor advised him to modify his life style and take

- A Beta-blocker,
- An ACE inhibitor,
- Lifelong low dose aspirin,
- Clopidogrel for1y and
- A statin
- Cardiac rehabilitation and life style modification

Explain the benefits of ACE inhibitors in Adel

They prevent cardiac remodeling following MI
They reduce after load and cardiac work.

The doctor advised him to take

- B-blockers,
- ACE inhibitors
- Statins

11- What is the mechanism of action of B blockers that could benefit Adel?

The β-adrenergic–blocking agents decrease the oxygen demands of the myocardium (due to ↓↓ cardiac work)

by blocking β1 receptors.

↓ heart rate

↓ cardiac contractility

↓ B.p.

β-blockers can be used with nitrates to increase

The doctor advised him to take

- B-blockers,
- ACE inhibitors
- Statins

What is the mechanism of action of statins that could benefit Adel?

What is the mechanism of action of statins that could benefit Adel?

CHD is correlated with elevated levels of (LDL-C); and triglycerides and low levels of (HDL-C)

Statins act by competitively inhibiting HMG-CoA reductase-the rate-controlling enzyme of the metabolic pathway that produces cholesterol in the liver

Depletion of intracellular cholesterol→ ↑ Number of LDL receptors on liver cells →

↓ LDL cholesterol in plasma, Plasma triglycerides

↑ Plasma HDL levels.

Statins has Pleotropic effects: improved endothelial function, reduced vascular inflammation,

Take home message

1. **Control of modifiable risk factors is a must:**

Control of hypertension
Weight reduction
Healthy diet and lipid-lowering drugs
Cessation of smoking

2. **Time is muscle:**

Patients should be educated to seek medical advice as soon as they experience chest pain
Cardiac muscle is **not** regenerated.
Hospitals should be equipped with primary PCI facilities, and PCI team should be available 24 hr

Take home message

3. The following **medications should be taken for life for patients of previous coronary syndromes:**

Anti-platetets

Beta blockers

Lipid-lowering drugs

4- Proper placement of **ECG leads**

Cholesterol lowering diet

Thank
you